breaking down and ulcerating. This man's face and body while this process was going on resembled those of a person suffering from small-pox. He was released after the condition had passed its worst and with good general health.

PART III.—TREATMENT.

In the jail all lepers were brought under excellent hygienic conditions. Good food, suitable clothing, enforced cleanliness, abundance of fresh air, and appropriate light labour were the lot of all. Sores and ulcers were kept clean and any little surgical measure deemed necessary was done. In addition to these general measures medicinal treatment of some kind was practised with most of the prisoners. The medicines tried were chaulmoogra oil, gurjun oil, salol, and salicylic acid ointment, each in selected groups. The conclusion come to was that none of these remedies was followed by any special benefit, and towards the end of the period of observation their use was abandoned for a simple symptomatic line of treatment. In December, 1906, and January, 1907, a trial was made of subcutaneous injections of 30 per cent. emulsions of iodoform in olive oil by which a German colonial officer, Dr. Diesing, was reported to have had success. Injections of four cubic centimetres of this emulsion were given to six selected cases. After 12 injections the practice was suspended to await results. No influence on the disease was detected up to the end of February, 1907. During the period of observation nine lepers died and 94 were released—22 with the symptoms alleviated, 21 with the symptoms more pronounced, and 51 practically in the same condition as on admission. In two of these which improved no symptoms could be found of the disease at all at release. In the majority of the cases it was recorded that the general health had improved during the stay in jail, this being not infrequently noted even in cases in which the special symptoms of leprosy seemed to have increased. Of the 94 released, 68 had gained in weight (average gain 9·46 pounds), 30 had lost weight (average loss 6·43 pounds), and six showed no change. The average duration of confinement of the 94 was 12·41 months. Of the nine cases who died only one was a maculo-anæsthetic leper. He was stated to have had the disease for 16½ years and had been in the leper jail for 13½ years. The others were mixed cases. The cause of death in all was some intercurrent affection—diarrhœa in six, tubercle of lungs in two, and gangrene in one. In all the leprosy was of many years' duration[18] and the prisoner was markedly debilitated by it.

# AORTIC ANEURYSMS; SUDDEN DEATHS; THE CAPACITY OF THE PERICARDIUM.

BY HERBERT A. SMITH, M.D. DURH., M.R.C.S. ENG., L.R.C.P. EDIN.

"I say that aneurysm is cured by rest and not by the surgeon!"—*John Hilton.*

THE mystery of sudden death commands interest, clinical, physiological, and forensic, and the last especially—the chemical, toxic, and traumatic causes—have a wide range of uncertainty, for the objective appearances of death do not always correspond with the classical modes thereof as given by Bichât, and with pathological causes there is often doubt. It is said by Virchow[1] "to be almost absolutely certain that the two most important kinds of death, asphyxia and paralysis, occur from overfilling of the right and left ventricles of the heart," yet in the following case both these cavities were found empty. Vivian Poore[2] says that sudden death obtains (1) by failure of blood, as in hæmorrhage, rupture of aneurysms, &c.; (2) failure of heart action through systemic, valvular, or thrombotic causes; and (3) failure of respiration through central or peripheral causes, that when the lungs cease first you get cyanosis and engorgement of the right heart and systemic veins, and when the circulation stops first you get pallor from a nearly empty left heart.

CASE 1.—On March 5th, 1908, I was summoned to a man in a fit. On arrival two minutes later he was dead; he had been pruning roses and suddenly feeling faint and giddy he fell to the ground. I found marked cyanosis, blueness of the lips and cheeks, and a congested venous state of the neck. The mouth was open, showing a protruding tongue, a condition suggesting some pressure on the venous return. Did the lungs cease before the heart? On making a necropsy (before an inquest) I found, after removing the front of the sternum and ribs to the subclavicular line, a distended viscus ready to burst; it was the pericardium pressing the diaphragm downwards and occupying a wide pulmonary area; on opening it 14 ounces of serum were baled out and then five ounces of soft black blood clot. The chambers of the heart, save a long thin clot in the right auricle, were empty and the valves were intact and competent, but an aneurysm of the ascending aorta (saccular) of the size of a walnut was found, the seat of rupture having a stellate appearance. The inner coat was stretched and torn and the other coats were soft, fatty, and friable. The intima right down to the thoracic aorta was riddled with areas of atheroma and calcareous plates and granules. There was no illness, no complaint, no lapse of work; yet the disease proceeded without let in a man of fine physique. It is said by Broadbent that "the favourite site of an aneurysm to rupture is through the unsupported sinuses of Valsalva," the intrapericardial portion of the ascending aorta, and that sudden death may occur without previous physical signs.[3] The lining of the pericardium common to the aorta and pulmonary artery in a single tube favours early rupture of the aneurysmal sac, yet with the atheromatous changes, the fact that no symptom is felt, no sign complained of, is noteworthy.

Some years ago a woman was admitted into the Middlesex Hospital for obscure pains in the chest and stabbing sensations, and gradually advancing pallor, faintness, and giddiness led to death. A post-mortem examination was made, and as the heart was taken out the pathologist pricked his finger by a needle which had traversed the œsophagus, trachea, pericardium, and the ascending aorta through a sinus of Valsalva, and the point of which was in the pericardium. Some 10 ounces of blood were found; trickling of blood alongside the needle had led to rupture and fatal flow.

Men live on the brink of a precipice. The cone of the fatal clot, the flimsy film, the flaw within the tube, and the fragile vessel or sac gives way without a signal.

CASE 2.—A city merchant whose life had been one of constant activity and excitement was under my care for aneurysm of the arch of the aorta for 11 years. During this time he presented most of the cardinal features of the disease and the saccular tumour grew to the size of a pear, eroding the sternum and extending the skin into a cone, yet strengthened by newly organised tissue. He kept his bed at first on and off for three months at a time for two years and took as much as two pounds of iodide of potassium with little fluid. He afterwards went daily to business, smoked two or three cigars, and enjoyed life. At 56 years of age he went for a winter cruise in the Mediterranean and coming home died somewhat suddenly from acute double pneumonia. The aneurysm had compressed behind the pulmonary artery and bronchial tubes and laterally had led to pulmonary condensation and absorption, thus favouring pneumonia.

CASE 3.—A few years ago I was summoned in the early morning to see a man, aged 56 years, who had fallen to the floor while entering a bath. I found him dead. There was marked pallor of the upper, and cyanosis of the lower, half of the face. A post-mortem examination elicited a thoracic aneurysm of the size of a tennis-ball which had absorbed a corresponding portion of lung and had ruptured into the posterior left mediastinum; it was saccular, occupying the costo-vertebral trough over the eighth and ninth ribs. The arterial opening was small and the site was favourable for support and for that side-flow or oblique eddy that favours coagulation. The right ventricle contained much blood clot. The sac wall was brittle, laminated, and lined with old and organising clot. Three months before Dr. T. T. Whipham had seen the case for backache, dyspnœa, and pleurodynia and had merely suspected an aneurysm, which had grown with great rapidity.

The early diagnosis of aneurysm by the x rays becomes important. Dr. David Arthur tells me that by its means aneurysms can be diagnosed from the merest budding to the point of rupture and that the time of giving way can be predicted with nicety outside the body. Moreover, by Hotznecht's process—the anterior right oblique position—the smallest aneurysm can be seen and shadowed, but the rate of

[18] Duration of disease not known in one case. Average duration of remaining eight cases was 10·94 years.

[1] Virchow: Post-mortems, p. 35.

[2] G. V. Poore: Medical Jurisprudence, p. 50.

[3] Broadbent: Heart Disease, p. 448.

growth hereby has yet to be determined. In a skiagram taken by Dr. D. Arthur but not published of Dr. Seymour Taylor's case[4] the space intervening between the heart and pericardium in the advance and retreat of the heart's action is clearly seen.

The normal amount of pericardial serum is that of lubrication. The pathological stretch of capacity of the pericardium, according to G. A. Gibson,[5] has reached eight pints of serum (cardiac). According to Jackson Clarke,[6] sufficient pus has been found in this viscus to mask the whole anterior pulmonary area of resonance, yet 18 ounces of blood sufficed in my case to render the pericardium taut. Such is the elasticity and capacity of the pericardium.

In spite of Dr. Taylor's strictures, my treatment of aneurysm medically would be rest (relaxed periodically), limited fluid, as much iodide of potassium and lactate of calcium as the patient could bear, and a prolonged stay at Woodhall, Sandrock, or Malvern—calm retreats whose spa waters are more or less rich in iodine, bromine, alum, and iron.

West Acton, W.

# THE PRECIPITIN REACTION IN HYDATID DISEASE.

BY D. A. WELSH, M.A., B.SC., M.D. EDIN.,
PROFESSOR OF PATHOLOGY IN THE UNIVERSITY OF SYDNEY, NEW SOUTH WALES;
AND
H. G. CHAPMAN, M.D., B.S. MELB.,
DEMONSTRATOR IN PHYSIOLOGY IN THE UNIVERSITY OF SYDNEY, NEW SOUTH WALES.

IN July, 1907, Fleig and Lisbonne[1] announced that they had succeeded in demonstrating the existence of a specific precipitin in the blood serum of an individual affected with a hydatid cyst and in the serum of animals immunised with hydatid products. They also emphasised the diagnostic significance of the reaction. Previous work[2] by Ghedini, by Joest, and by Gherardini had yielded contradictory results. With the more abundant material at our disposal we have been able to corroborate and to supplement the main observations of Fleig and Lisbonne in regard to hydatid invasions in the human subject. We have found that when a sufficiency of blood serum is mixed with a suitable hydatid fluid and the mixture allowed to stand for 18–20 hours at the room temperature, a well-marked precipitate never fails to appear if the serum be taken from a patient infected with hydatids (nine cases), and that precipitation does not occur when the serum is taken from persons not so infected (four suspected cases and five healthy controls). In the four suspected cases examined by us a negative reaction was obtained, and at operation in each case the absence of hydatid invasion was revealed. These results are so consistent, though as yet few in number, that they encourage us in the belief that in the precipitin test for hydatid invasion we have a reliable means of diagnosis. We find, however, that certain conditions are essential to the satisfactory performance of the test.

In regard to the serum (antiserum) developed in a hydatid patient, our experience has been that, with few exceptions, it is characterised by a low precipitable content. Hence, as noted by Fleig and Lisbonne, about 12 drops of the patient's serum are usually required to yield a satisfactory amount of deposit. In a previous paper[3] dealing with precipitin antisera prepared in the rabbit by injections of alien blood serum or of egg-white, we noted that two drops (0·1 cubic centimetre) of the antiserum were usually sufficient to yield a well-marked precipitate. We were also led to the conclusions that the precipitate was derived mainly from the antiserum and that different antisera might differ greatly in their precipitable content.

Fleig and Lisbonne observed that the anti-substances in the serum of a hydatid patient may gradually disappear after operation. That this disappearance of the precipitin is not necessarily associated with removal of all the cysts is shown by one of our cases. In a man, aged 42 years, with multiple hydatid cysts of the liver (not all of which could be removed at operation) we found a well-marked reaction before operation, but at the end of the sixth week and again at the end of the seventh week after operation the serum yielded only a minute trace of deposit. The patient died in the eighth week after operation, having become rapidly weaker and more emaciated, and it may be that in these circumstances his capacity for reaction was failing. On the other hand, a girl, 14 years old, with multiple hydatids of the peritoneum following a ruptured hydatid of the liver gave a well-marked reaction eight weeks after operation. She was well nourished and was leaving the hospital apparently cured. The indication, however, was that she was still infected with hydatids and that the tissues were reacting to their presence.

In regard to the hydatid fluid, our practice has been to pass it through a Chamberland filter. We have obtained more abundant precipitates with one cubic centimetre hydatid fluid than with two cubic centimetres (as recommended by Fleig and Lisbonne), probably because the small amount of the homologous protein contained in the hydatid fluid is sufficient to produce a marked precipitation in the antiserum, while the interaction is not hampered by over-much dilution. This also is in harmony with our previous work on serum and egg-white precipitins.

In the course of our experiments with hydatid precipitins we encountered one hydatid fluid which gave no precipitate, in addiments of two cubic centimetres, either with the serum of the patient from whom it was obtained, or with the serum of other hydatid patients. When, however, these tubes, after standing 48 hours without showing any precipitate, each received the further addition of one cubic centimetre of clear fluid from another hydatid invasion, distinct precipitates were obtained. It was certainly anomalous to find that the first hydatid fluid failed to precipitate the serum of the patient from whom it was obtained, while the serum of that patient reacted strongly with the hydatid fluid obtained from a different patient. We may note that each of the hydatid fluids above referred to contained, approximately, the same amount of albumin (about 0·025 per cent.).

The fact that not all hydatid cysts contain fluid capable of interacting to give a precipitate with the serum of the patient may be an explanation of the negative results obtained by earlier workers. What we would emphasise is that hydatid fluids may vary greatly in their capacity for interaction with the serum of a hydatid patient, but that a hydatid fluid which reacts strongly with the serum of the patient from whom it was obtained reacts also strongly with the serum of other hydatid patients. These facts are of importance in the selection of a stock of hydatid fluid in the diagnostic applications of the test.

*Summary of cases of hydatid invasion, all of which gave a marked precipitate reaction when tested before operation.*—1. Male, 35 years. Three small hydatid cysts with clear contents in peritoneum. Hydatid of liver removed eight years ago. 2. Male, 42 years. Multiple hydatid cysts of liver, some of which were full of clear fluid, others shrunken and inspissated. Not all cysts removed, yet reaction very feeble six and seven weeks after operation. Death in eighth week. 3. Male, eight years. Large single cyst of liver with clear contents. 4. Female, 14 years. Multiple hydatids of peritoneum with clear contents, following ruptured hydatid of liver. Marked reaction eight weeks after operation. 5. Male, 13 years. Single hydatid cyst of liver with clear fluid contents. 6. Female, 17 years. Multiple cysts of peritoneum with clear fluid contents, dense matted adhesions. 7. Male, 50 years. Suppurating hydatid cyst in muscles of thigh, well-marked reaction three weeks after operation. 8. Male, 48 years. Large hydatid cyst of liver with slightly turbid contents. 9. Male, 23 years. Large hydatid cyst of liver with slightly turbid contents.

*Conclusions.*—1. The interaction between selected hydatid fluids and a sufficiency of the serum of a patient affected with hydatid disease has, in our experience of nine cases, never failed to give a positive precipitin reaction when tested before operation. 2 Not all hydatid fluids are capable of eliciting this reaction, and their failure is not associated with any noticeable diminution of their protein content. 3. Persistence of a marked reaction some weeks after operation probably indicates the continued presence of the parasite, but the disappearance of the reaction does not necessarily indicate complete removal of the cysts. 4. A positive reaction is independent of the site of the hydatid cyst (hepatic, peritoneal, muscular) and independent also of the nature of its contents (clear, turbid, or purulent).

---

[4] THE LANCET, March 7th, 1908, p. 702.
[5] G. A Gibson: Textbook of Medicine, vol. ii., p 52.
[6] Jackson Clarke: Surgical Anatomy, p. 303.
[1] Comptes Rendus de la Société de Biologie, tome lxii., No. 23, 1907, p. 1198
[2] Published in 1906 and quoted by Fleig and Lisbonne, loc. cit.
[3] Welsh and Chapman: Proceedings of the Royal Society, B. vol. lxxviii., p. 297, 1906.